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Tommaso Castroflorio,[1] Andrea Bargellini,[1,2] Gabriele Rossini, [1,2]

Giovanni Cugliari, [3]Andrea Deregibus,[1,2] Daniele Manfredini[4]

[1]Department of Surgical Sciences, Specialization School of Orthodontics, Dental School, University of Torino, Via Nizza 230, 10126 Torino, Italy

[2]Department of Surgical Sciences, Gnathology Unit, Dental School, University of Torino, Via Nizza 230, 10126 Torino, Italy

[3]Department of Brain and Behavioural Sciences, Unit of Medical and Genomic Statistics, University of Pavia, Strada Nuova, 65, 27100, Pavia, Italy

[4]TMD Clinic, Department of Maxillofacial Surgery, University of Padova, Via Giustiniani 2, 35122 Padova, Italy

Corresponding author:

Dr . Andrea Bargellini

Via Nizza 230, 10126 Torino, Italy

Tel 0039 3339233219

bargelli@ipsnet.it

Abstract

Background

Over the years, several strategies were proposed to diagnose sleep bruxism. The aim of this study was to compare clinical SB diagnosis with the instrumental diagnosis of SB obtained with a device providing EMG/ECG recordings.

Methods

45 subjects (19 men and 26 women, mean age 28 ± 11 years) were selected among patients referring to the Gnathology Unit of the Dental School of the University of Torino. An expert clinician confirmed a clinical diagnosis of SB when one or more signs/symptoms were present. Furthermore, all participants underwent an instrumental recording at home with a portable device (Bruxoff®, OTBioelettronica, Torino, Italy) allowing a simultaneous recording of EMG signals from both the masseter muscles as well as heart frequency. Statistical procedures were performed with the software Statistical Package for the Social Science v. 20.0 (SPSS 20.0®, IBM, Milan, Italy).

Results

Based on the Bruxoff software analysis, 26 subjects (11 males, 15 females, mean age 28 ± 10 years) were diagnosed as sleep bruxers, whilst 19 subjects (7 males, 12 females, mean age 30 ± 10 years) were diagnosed as non-bruxers. The correlation value between the clinical and EMG-ECG SB diagnoses was low (ϕ value = 0.250), with a 62.2% agreement (28/45 subjects) between the two approaches (kappa = 0.248). Assuming instrumental EMG-ECG diagnosis as the standard of reference for definite SB diagnosis in this investigation, the false-positive and false-negative rates were unacceptable for all clinical signs/symptoms

Conclusions

Findings from clinical assessment are not related with SB diagnosis performed with a portable EMG-ECG recorder.

Keywords: Sleep bruxism; bruxism; clinical criteria; diagnosis.

Introduction

Sleep bruxism (SB) is a sleep-related motor disorder characterized by involuntary phasic (rhythmic) or tonic (sustained) motor activity in the masticatory muscles (e.g. masseter, temporalis) during sleep. It can be associated with a number of clinical problems, including orofacial pain, tooth wear and failure of dental restorations.[1,2] Furthermore, it is considered a risk factor for complications in implant- and teeth-supported rehabilitations.[3,4]

Over the years, several strategies were proposed to diagnose bruxism.[5,6] The literature showed that the wide majority of data came from studies adopting a self-reported bruxism detection,[7] which is suitable, at best, to indicate a "possible" bruxism. Such an approach is in contrast with the proposed standards of reference for SB diagnosis, which require definite measurements by means of polysomnography (PSG).[8] In particular, PSG studies have shown that SB features a typical muscular activation pattern (i.e., rhythmic masticatory muscle activation [RMMA]), which is usually associated with arousals of autonomic nervous system.[9-11]

Nonetheless, PSG has some disadvantages, such as the high cost, the amount of time needed for manual/visual scoring, the laboratory setting, not providing information of oral behaviors occurring in home environment, and the potential for bias due to the examiner's skill.[12] Thus, it is mainly used for research purposes and has a minor impact on the clinicians' daily routine.[13]

For such reasons, the need for introducing easy-to-use strategies for SB diagnosis and measurement has recently been advocated.[13] Recent studies have validated a portable device providing combined surface electromyography (EMG) and electrocardiography (ECG) measurements, which showed an excellent diagnostic accuracy with respect to PSG for the diagnosis of SB.[14-16]

Despite such efforts, SB diagnosis in the clinical setting is still mainly based on clinical assessment.[10] Unfortunately, a recent consensus panel suggested that such clinical diagnosis should be able to detect, at best, "probable" bruxism.[8] Also, it was recently pointed out that data are absent as far as the relationship between such clinically-diagnosed and instrumental/PSG-diagnosed bruxism is concerned.[17]

Based on these premises, the aim of this study was to compare the "probable" bruxism diagnosis based on the clinical assessment with the instrumental diagnosis of SB obtained with a device providing EMG/ECG recordings. The study design aimed to answer the clinical research questions: "is there a correlation between the clinical and instrumental SB diagnosis?" The null hypothesis was that purported clinical signs and symptoms of SB (i.e., transient jaw muscle pain in

the morning, muscle fatigue at awakening, presence of tooth wear or shiny spots on restorations) are not related with instrumentally diagnosed SB. If the null hypothesis was rejected, the diagnostic value of clinical SB diagnosis could approximate the needed requirements for a "definite" diagnosis, thus having potentially relevant clinical implications.

Materials and Methods

Subjects and study design

The study was performed on 45 subjects (19 men and 26 women, mean age ± standard deviation (SD) 28 ± 11 years) selected among patients referring to the Gnathology Unit of the Dental School of the University of Torino. To ensure that individuals with different SB severity took part to the study, participants were initially recruited based on a clinical assessment suggesting their probable bruxism (N=22, 10 males and 12 females, mean age ± SD 26 ± 4) or the absence of bruxism (N=23, 9 males and 14 females, mean age ± SD 32 ± 14). Exclusion criteria were: 1) presence of extensive prosthodontic rehabilitations, 2) missing teeth, with the exception of the third molars 3) periodontal disease, 4) Presence of temporomandibular joint disorders, as diagnosed with the Research Diagnostic Criteria for TMD,[18] 5) medical history of neurological, mental, or sleep disorders (e.g., periodic leg movements, insomnia). Furthermore, the subjects were not under medications at the time of recording, and were not under the effect of alcohol, nicotine or caffeine.

An expert clinician based on the presence of the following diagnostic criteria made the clinical assessment for SB: transient jaw muscle pain in the morning, muscle fatigue at awakening, presence of tooth wear or shiny spots on restorations. Based on that, a clinical SB diagnosis was assigned when one or more of the above clinical signs/symptoms were present.

All participants underwent an instrumental recording at home with a portable device (Bruxoff®, OTBioelettronica, Torino, Italy) allowing a simultaneous recording of EMG signals from both the masseter muscles as well as hearth frequency. The three signals were sampled at 800 Hz, with 8 bit resolution. Data were stored on a MicroSD card as a binary file. The EMG channels were filtered between 10 and 400 Hz with a gain of 4300. The ECG channel was filtered between 15 and 160 Hz with a gain of 700. Masseter muscles' EMG activity was detected with disposable bipolar AgCl concentric electrodes (Code®, Spes Medica, Battipaglia, Italy), with a 16 mm radius. The

choice of adopting such electrodes was due to their easy applicability and design, avoiding muscle fiber electrode orientation problem and reducing EMG crosstalk.[19,20] ECG recordings were detected with a disposable bipolar electrode located on the left side of the thorax, at about 5-10 cm below the sternum. Each participant underwent two consecutive recording nights (at least 4 hours of sleep per night). The first night was an accommodation session to familiarize with the device, and only data recorded during the second night were considered for statistical analyses. The recording procedure provided that five tapping movements before sleep and after getting up in the morning were performed, in order to easily recognize the beginning and the end of the recordings. After the five tapping movements at the beginning of the recording session, the subjects performed three maximum voluntary clenching (MVC) on teeth. The clenches should last 3 sec each and be separated by a 10-sec rest. The greatest MVC value was used to normalize the EMG values as a percent of MVC. Masseter EMG bursts with duration exceeding 0.25s were selected for oromotor activity scoring.[9,21]

Previous studies showed a high sensitivity and specificity of the portable device (92.3% and 91.6% respectively) when the diagnostic cut-off was set at 4 SB episodes per hour,[14] as suggested by published criteria.[22] Furthermore, a reliability study showed a good reproducibility as far as the number of SB episodes per night, SB episodes per hour, and heart frequency are concerned.[15] Based on that, the Bruxoff software (Bruxmeter software®, OTBiolettonica, Torino, Italy) was set to score automatically the presence of SB events based on the following features: mean masseter EMG amplitude at least 10% of maximum voluntary clenching activity, preceded (1-5 seconds interval) by an approximately 20% increase of heart rate (beginning 1 second before RMMA onset).[9,22] Oromotor activity during wakefulness before falling asleep was excluded from scoring.

The procedures were approved by the Lingotto Dental School ethic committee. All individuals gave their informed consent in accordance with the Helsinki Declaration and understood that they were free to withdraw from the experiment at any time.

Statistical Analysis

The design of statistical analyses aimed to answer the underlying clinical research question of the study, viz., assessing the correlation between clinical and instrumental bruxism diagnosis.

The frequency of presence of the various clinical signs/symptoms as well as of a positive SB diagnosis with the Bruxoff device was described. Contingency tables were created to compare the Bruxoff findings (columns) and the clinical variables (rows). The correlation between the clinical

findings and the instrumental diagnosis was assessed by means of ϕ coefficient, which is a measure of the degree of association between two binary variables. Such coefficient is similar to the correlation coefficient in its interpretation: ϕ values range from -1.0 to + 1.0, indicating different levels of negative or positive correlation. As a general rule for correlation analyses, values higher than 0.7 are considered supportive of a strong positive correlation.[23]

In addition, a T-test for unpaired samples was performed to compare the mean SB index, as derived with the Bruxoff device, of subjects having or not having the various clinical findings.

All statistical procedures were performed with the software Statistical Package for the Social Science v. 20.0 (SPSS 20.0®, IBM, Milan, Italy). For each analysis a p-value<0.05 was set.

Results

Based on the Bruxoff software analysis, 26 subjects (11 males, 15 females, mean age 28 ± 10 years) were diagnosed as sleep bruxers, whilst 19 subjects (7 males, 12 females, mean age 30 ± 10years) were diagnosed as non-bruxers. The correlation value between the clinical and EMG-ECG SB diagnoses was low (ϕ value = 0.250), with a 62.2% agreement (28/45 subjects) between the two approaches (kappa=0.248) (Table 1).

The frequency of positive clinical items in the study sample ranged between 31.1% for facial pain and muscle stiffness and fatigue at awakening to 42.2% for tooth wear or shiny spots on restorations, and up to 53.3% for masseter hypertrophy. The correlation values with SB were low for each clinical sign/symptom, ranging from ϕ=-0-045 to 0.196 (Table 2). This means that, if a clinical SB diagnosis was based on any single specific clinical sign/symptom, the agreement with instrumental SB ranged between 46.6% (21/45 subjects) for muscle stiffness at awakening and 60% (27/45) for masseter hypertrophy. Assuming instrumental EMG-ECG diagnosis as the standard of reference for definite SB diagnosis in this investigation, the false-positive and false-negative rates were unacceptable for all clinical signs/symptoms (Table 3).

The average SB index was different between subjects having or not having masseter muscle hypertrophy (p=.033), whilst there were not any significant differences for the other clinical signs/symptoms, with p-values ranging from .351 to .645 (Table 4).

Based on the above, the null hypothesis that purported AASM clinical signs and symptoms of SB (i.e., jaw pain, masseter muscle hypertrophy, tooth wear or shiny spots on restorations,

morning stiffness in the jaw muscles) are not related with instrumentally diagnosed SB could not be rejected.

Discussion

The aim of this study was to compare the clinical diagnosis of SB, viz., a so-called "probable" bruxism, with SB diagnosis based on EMG-ECG recordings obtained with a validated portable device.

Over the years, several clinical signs and symptoms have been proposed as markers of SB. They include, among the others, the presence of transient jaw muscle pain in the morning, a feeling of fatigue or stiffness in the jaw muscles at awakening, abnormal tooth wear, and masseter muscles' hypertrophy. Despite it was suggested that none of these signs and/or symptoms may represent a direct proof of ongoing SB,[5] their presence is still considered suggestive of a clinically diagnosed bruxism.[8,10]

Results clearly indicated that the above clinical criteria, which were selected based on literature suggestions, do not correlate with an instrumental SB diagnosis. Indeed, none of them was significantly related to an instrumental diagnosis of ongoing SB, with the minor exception of a higher SB index in subjects with masseter hypertrophy. This implies that the resulting clinical diagnosis had a very poor agreement ($k = 0.248$) with the definite SB diagnosis, as obtained with devices measuring EMG activity. Of course, it must be remarked that, for an actual diagnosis of definite SB to be made, full PSG recordings should have been required, but their adoption is unlikely to change the study findings. Indeed, the EMG-ECG recorder adopted in this investigation showed an excellent correlation with PSG findings in a previous study,[14] and was thus introduced in the research setting to ease data gathering.

Despite seemingly discouraging, data from this investigation are actually in line with the fragmental literature on the relationship of SB with pain and tooth wear. In general, the literature suggested that the proposed PSG cutoff values for SB were suitable for discriminating between patients with and without tooth wear,[24] whilst they were not suitable to intercept subjects who are at risk for developing pain in the jaw muscles.[25-29]

Our findings are open to interesting considerations. Indeed, at a first glance it could be concluded that a clinical SB diagnosis is not acceptable, so that even the recently defined

"probable" bruxism is far from being "probable". On the other hand, despite quantitative recordings are without any doubts the standard requirement for a definite sleep bruxism diagnosis, it emerged that several issues need to be clarified concerning the interpretation of bruxism measurements. Indeed, it seems that neurologically driven criteria drawn from PSG studies are not related with the clinical consequences of SB, especially as far as muscle fatigue and pain are concerned. A possible explanation for such lack of relationship is that EMG adaptations to pain in the jaw muscles may lead to a reduced muscle activity (i.e., less SB) in patients with pain.[30-32] This means that even those types of bruxism activities (e.g., prolonged, high intensity, isometric contractions such as in the case of mandible thrusting) that are plausible risk factors for muscle pain are likely to be detected as such only in the early stages of pain onset, before protective adaptations turn in to reduce muscle activity.[13]

A recent review suggested how to refine some concepts underlying a potentially "ideal" SB diagnosis.[13] Based on that, this study's findings support the view of SB as a variegate motor phenomenon, and not as a disorder per se. Thus, until the different motor activities that are currently grouped together under the umbrella term "bruxism" are not properly discriminated based on their EMG features, it is unlikely that we are able to get deeper into the clinical picture.

In short, taken together, our findings suggested that currently-proposed clinical diagnostic criteria for SB are not evidence-based.

Conclusions

This study showed that findings from clinical assessment are not related with SB diagnosis performed with a portable EMG-ECG recorder. Further studies on larger and more representative samples are needed to get a deeper insight to the relationship between an instrumental SB diagnosis and the purported clinical signs/symptoms.

Acknowledgments

The procedures were approved by the Lingotto Dental School Ethics Committee. All individuals gave their informed consent in accordance with the Helsinki Declaration. The research funding was provided by University of Torino.

Conflict of interest

The authors do not have any financial conflicts of interest or relationship with any financial organization that may be interested in the contents of this manuscript.

The authors declare that all them have contributed to conceptualize and perform the investigation as well as to manuscript's writing and revision before submission.

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Tables

Table 1. Cross-tabulation of SB diagnosis based on either clinical or EMG/ECG findings.

		EMG/ECG diagnosis	
		No SB	SB
Clinical diagnosis	No SB	14	7
	SB	10	14

Table 2. Prevalence of the different clinical signs/symptoms in subjects with or without EMG/ECG diagnosed SB, and levels of correlation with SB.

	Prevalence in SB (%)	Prevalence in non-SB (%)	Correlation value (ϕ)
Transient jaw muscle pain in the morning	33.3	28.6	0.051
Muscle fatigue at awakening	29.2	33.3	-0.045
Tooth wear	41.7	42.9	-0.012
Masseter hypertrophy	62.5	42.9	0.196

Table 3. False-positive and false-negative SB findings based on the presence of clinical signs/symptoms, and their agreement with SB diagnosis.

	False-positive SB findings (%)	False-negative SB findings (%)	Agreement (%)
Transient jaw muscle pain in the morning	42.9	51.6	51.1
Muscle fatigue at awakening	50.0	54.8	46.6
Tooth wear	47.4	53.8	48.8
Masseter hypertrophy	37.5	42.9	60

Table 4. SB index of subjects with and without the different clinical signs/symptoms.

	SB index of positive subjects (%)	SB index of negative subjects (%)	P-value
Transient jaw muscle pain in the morning	5.0±3.4	4.1±2.8	.351
Muscle fatigue at awakening	4.1±3.1	4.5±3.0	.645
Tooth wear	4.8±3.6	4.08±2.5	.431
Masseter hypertrophy	5.2±3.4	3.3±2.1	.033